Observations

ON
THE SIGNIFICANCE OF RETINAL
HAEMORRHAGES.*

BY

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As, in a generous but perhaps unduly sanguine mood, the Master of the Congress and his colleagues have placed in my hands the responsibility of opening this discussion, I must conclude that in their judgement it will be an advantage to have a statement of the problems affirmed by the existence of retinal haemorrhages as these present themselves in the practice of a physician. The issue arises in two different circumstances: (1) when, in consequence of the discovery of one or more haemorrhages in the retina, there appears the question whether these are of purely local significance or whether, on the contrary, the haemorrhage is an announcement of some diseased condition either affecting the body generally or situated in some more or less remote organ; and (2) when, the patient's health being in one fashion or another admittedly unsatisfactory, the recognition of retinal haemorrhage imposes itself as an additional fact, and one that demands consideration in the discussion of the diagnostic and prognostic aspects of the case. In the one instance the question is, Is there, in addition to the retinal haemorrhage, any other evidence of disease, and if so, what is the relation between the two observations? In the other, What aid or influence does the retinal haemorrhage contribute to the clinical interpretation of the facts taken as a whole? Both in the one position and in the other it is obvious that a retinal haemorrhage in its clinical meaning can be judged only when all the facts—positive and negative—relative to the individual patient have been duly collected and studied; in other words, it is a claim for nothing less than a complete and thorough clinical examination. Not that in this respect retinal haemorrhage stands in a peculiar or exceptional position. On the contrary, it comes under the general rule that, important as an examination of individual organs may be, still more important is the examination of the patient.

The first question in our discussion must necessarily be, May retinal haemorrhage exist without clinical significance? The answer, I think, must be in the affirmative. That is, routine ophthalmoscopic examination now and again discovers retinal haemorrhage in quite exceptional circumstances; and the circumstances are so various, and the event itself so occasional, that it seems impossible to attach to such haemorrhages any precise meaning and value. In my own notes I find records of retinal haemorrhages—few in number and slight in degree—in individual cases of pneumonia, pyonephrosis, malignant disease of the viscera, mitral disease, and ill-defined clinical conditions that had to be content with such an unsatisfactory label as "anaemia." As the routine use of the ophthalmoscope becomes more general under the influence of the conviction that inspection of the fundus oculi is an essential part of every complete clinical examination, it may be anticipated that unexpected retinal haemorrhages in various circumstances will be a more familiar experience.

In a second group of retinal haemorrhages the significance is obvious. I refer to haemorrhages due to violence. Of the consequences of direct violence applied to the eyeball or to the skull I say nothing, as such cases have for many years been outside my experience; nor do I know anything directly of the retinal haemorrhages said to be frequently found in newborn infants. My reference is to what may be called general bodily violence or physical shock without evidence of injury to the head. Retinal haemorrhages may be observed in these cases, and they have this practical aspect—that as the defect of sight to which they may give rise may be postponed or delayed, the responsibility of the violence for this defect is apt to be contested unless the fundus oculi has been examined in an early stage of the case. The experience is one more argument for the routine use of the ophthalmoscope even when no local claim for this procedure seems to exist.

As a third group, I mention retinal haemorrhages so severe as obviously to endanger vision, yet not provoked by violence, and not accompanied by signs of organic disease in other parts of the body. The most characteristic members of this group fall into the class described by Mr. Eales of Birmingham in 1880; the patients, lads or young men, the victims of severe, perhaps recurrent, retinal haemorrhage in one or both eyes, but otherwise apparently healthy though often giving a history of more or less extreme epistaxis. Upon cases of this order I propose to offer three remarks.

First, in Mr. Eales's original records every patient is said to have shown a "trace," or "a faint trace," of albuminuria. Happily no individual observer, I take it, sees many of these cases, but in my own experience, limited in this respect as it is, I have not detected albuminuria, and I suggest the point as one upon which members might give the Congress the benefit of their observations.

Secondly, Mr. Eales described in the retina, in cases that he named examples of "temporary functional albuminuria," "white specks . . . presumably from previous haemorrhages," the patients being males of 11 to 28 years of age. Now albuminuria in apparently healthy persons is a problem often presented to the physician, the question being, Is the albuminuria an expression of mere functional disturbance or is it a signal of organic renal disease? Personally I should say that one of the notes of functional albuminuria is the absence of retinal change, and retinal haemorrhages or "white specks . . . presumably from previous haemorrhages" would make me hesitate at so innocent a diagnosis. There must be, I imagine, many members of the Congress who have been asked to report on the ophthalmoscopic facts in case of albuminuria of more or less uncertain significance, and experiences gathered in this direction would, I am sure, be a valuable contribution to the present discussion.

My third remark is: If the essential note of Eales's disease is retinal haemorrhage unprovoked by violence and unaccompanied by evidences of organic change, then it will be very difficult to confine this name in any scheme of logical classification either to youthful patients or to the male sex. For, undoubtedly, retinal haemorrhage, strictly under the conditions just defined, occurs in middle-aged and even in elderly persons; and some of the victims are women. In other words, while the most extreme and disastrous examples of what may be called spontaneous or unexplained retinal haemorrhages are found in young men, this fashion of haemorrhage is not restricted to the earlier years of life, nor is it confined to the male sex. If a line of division is to be drawn between true cases of Eales's disease and other cases of apparently spontaneous retinal haemorrhage, I ask members of the Congress to say exactly where it is to be drawn. The difficulty is a real one. It is illustrated, to take but a single example, in a paper written some years ago by Mr. Leslie Paton and Dr. Erasmus Paramore, where, in a short series of unexplained haemorrhages, the writers include two women, one aged 27 years and the other 34 years. The point is one that seems to invite discussion.

At first sight it might appear that uncomplicated and unexplained retinal haemorrhage forms purely an ophthalmological problem. But really it is not so. On the contrary, it occupies merely one page of a fairly considerable chapter. For haemorrhages apart from violence, and unaccompanied by appreciable organic change, occur in almost all parts of the body.¹ Epistaxis, for example, without nasal lesion is so well known that its occurrence is little more than a mildly exciting domestic incident; haematemesis does not always mean ulceration of the gastric or duodenal mucous membrane; there are cases of haematuria, not infrequently recurrent haematuria, in which no structural alteration can be found in the urinary passages; even haemoptysis sometimes stands in a similar enigmatic position; and cerebral and cerebellar haemorrhages in adolescents are by no means altogether unknown. Thus retinal haemorrhages existing as an isolated clinical event have their parallels if not their explanation. An explanation is indeed difficult. So far as I have followed the discussion the key seems to have been sought in one direction, and in one direction only—namely, in an attempt to determine an alteration in the coagulation rate of the blood. The results have not been encouraging, for, while on one side it is announced that coagulation is hastened, we hear from other quarters that coagulation is delayed; and the confusion is increased by a

* Introduction to a discussion at the Oxford Ophthalmological Congress, July 6th, 1922.

¹ See Sir Wm. Hale-White, *Lancet*, 1912, i, p. 416; and Professor George R. Murray, *British Medical Journal*, 1913, ii, p. 1521.

third statement to the effect that no departure from the normal can be appreciated. With all respect I dare to say that even were this point finally and authoritatively settled we should still be without an explanation of the haemorrhage. For, while the readiness, or the difficulty, with which the blood coagulates must be a large influence in determining the volume or duration of a haemorrhage, no modification in either of these directions will account for the actual occurrence of haemorrhage. To produce this there must be rupture of vessels, and rupture of vessels, in the absence of violence and of increased intravascular stress—and neither of these factors is in issue here—can only follow changes (nutritional or structural) in the vessel walls. It is therefore in the direction of agencies calculated to damage the walls of the vessels that an explanation of these quasi-spontaneous retinal haemorrhages must be sought. There are here two considerations which may appropriately be borne in mind.

First, it is beyond doubt that the walls of the finer blood vessels are particularly susceptible to the influence of pathological changes in the blood quality; and it would seem that in at least many of these changes each has its special or selected area of mischief. Thus in typhus we get haemorrhages in the skin; in scurvy, in the gums; in Henoch's purpura, in the intestinal wall; in influenza, epistaxis; and in cantharides poisoning, haematuria. Evidently in each of these instances the blood vessels in a particular area have been damaged by a poison that has to the affected vessels a specific pathological relationship. Is it not probable that in similar fashion the retinal blood vessels, or the retinal blood vessels in some persons, may be damaged by modifications of the blood quality that are at present beyond our power of detection and recognition?

A second influence possibly related to these spontaneous retinal haemorrhages is that the blood vessels in different persons possess different degrees of fragility. In some a mere friendly grasp of the arm is followed by ecchymosis, while in others a much greater degree of violence is necessary to produce a bruise. Once again, may it not be that there are individuals in whom the retinal vessels are unduly fragile as the result either of an inherited peculiarity or of an acquired defect, and that upon such vessels minor and perhaps unperceived degrees of violence act with disastrous consequences?

Summing up, therefore, this part of the discussion, I submit the following propositions: That retinal haemorrhages existing as an isolated clinical fact are strictly parallel to other haemorrhages which occur in various parts of the body apart from obvious violence and without appreciable organic lesion; that a departure, even if it exists, from the normal rate of blood coagulation will not account for these haemorrhages; and that the probable explanation is rupture of vessels following damage to their walls, this damage in turn resulting either from some subtle change in the blood quality or from slight degrees of violence—perhaps mere changes in intracellular tension—acting on vessels which for one reason or another are particularly susceptible to the destructive influence of mechanical forces. Possibly it may be suggested that the event immediately antecedent to the haemorrhage is thrombosis; and this may well be true. But thrombosis in its turn requires an explanation, and it may be doubted whether intravascular blood-clotting ever occurs in vessels which possess a healthy and undamaged endothelial lining. So that once again there is need for prior damage, nutritional or mechanical, to the vessel wall. Given this, either direct rupture is conceivable, or thrombosis, this latter establishing circulatory conditions which mean a breach in the integrity of the vessel and the escape of the fluid contents into the neighbouring tissues. In a word, whether the rupture immediately responsible for the haemorrhages is direct or is secondary to thrombosis, there is necessary as an antecedent event some preliminary prejudice to the anatomical or physiological values of the vessel wall.

I now propose to find support for the order of events just suggested—that is, damage to the vessel wall, thrombosis, rupture, haemorrhage—in the retinal haemorrhages (a well-known class) consequent on thrombosis of the central retinal vein. I have had the opportunity of examining for my surgical colleagues a considerable number of cases of this order, and with three exceptions (to be mentioned later) the patients have been elderly—50 years and upwards—and have exhibited evidences, and often conspicuous evidences, of cardio-vascular degeneration—thickened and deformed arteries, high sphygmometer readings, left cardiac hypertrophy, and sometimes albuminuria, though rarely convincing signs

of nephritis. Here, then, we have manifest and undeniable evidence of structural alterations in the vascular apparatus, and even apart from actual pathological demonstration—and this is by no means wholly wanting—there is a moral certainty that with evident arterial degeneration changes of a similar order will be present in the walls of the veins. Upon such damaged areas clot formation may readily be implanted, with, as a natural consequence, blocking of the circulatory movement, further prejudice to the vessel wall, rupture, and haemorrhage.

It may, however, be said that the argument here presented cannot apply to the occasional cases of thrombosis of the central retinal vein occurring in young persons free from obvious vascular disease and offering normal or approximately normal sphygmometer readings; and it must be admitted that in such cases we cannot claim the overt evidence of vascular disease seen in elderly persons affected by retinal venous thrombosis. On the other hand, it is certain that young persons are not altogether free from the risk of thrombosis, as is seen, for example, in the case of chlorotic girls. Such thrombosis must be either primary—that is, spontaneous—or secondary to changes in the vessel wall at the site where the clot formation takes place. But spontaneous intravascular blood-clotting is, for reasons already given, highly unlikely. Hence it seems necessary to admit that even when objective evidence of change in the vascular structures is wanting the occurrence of thrombosis implies that such change as a fact has actually taken place. In other words, retinal venous thrombosis, in the younger patients as in the elderly, is secondary to some fashion of alteration in the vessel wall. My suggestion, therefore, is that such cases form, as it were, a connecting link between the group of unexplained haemorrhages already considered and the cases of retinal venous thrombosis as this is more commonly seen in elderly patients. In all, I assume as the primary event pathological changes in the vessel walls, and following these changes either direct rupture or thrombosis prior to rupture. The groups, however, differ from one another in the site of the preliminary vascular damage and in the degree of clinical evidence available for the recognition of this damage.

I now pass to retinal haemorrhages—other than those consequent on retinal venous thrombosis—found in association with high sphygmometer readings. Personally I speak of “high sphygmometer readings” rather than of “high blood pressures,” for the sphygmometer reading is a fact, whilst its translation into terms of blood pressure involves an inference, and an inference, moreover, which has not wholly escaped an accusation of inaccuracy. If in scientific terminology names having a descriptive quality are selected, clearly they should be descriptive of facts and not of opinions about facts. Our immediate point, however, is the significance of retinal haemorrhages in patients yielding high sphygmometer readings. This group is a fairly considerable one, but its constituent members differ widely from one another both in character and in outlook. At one extreme are cases of renal disease without dropsy—the so-called small white kidney—occurring in young adults, often marked by epistaxis, and leading almost invariably in the course of a few months to a fatal issue. At the other extreme is a single haemorrhage in, say, a middle-aged or elderly patient, possibly destructive of central vision in the affected eye, but, as proved by the subsequent course of events, quite consistent with health and activity prolonged over a considerable term of years. And between these two extremes lie various degrees and gradations of arterial change and various degrees and gradations of renal mischief. In either extreme, as well as in the intermediate cases, retinal haemorrhage may be the first event to attract attention, or the haemorrhage may be found on examination, although the patient has not recognized any visual disability. Does the occurrence of retinal haemorrhage render any substantial assistance in the differential diagnosis or in the prognosis of the members of this large and varied group? I doubt it. Guidance in these directions is to be sought, not in the presence or the absence of retinal haemorrhage but in the state of the heart, the condition of the arteries, and in the efficiency or non-efficiency of the renal excretory functions.

In connexion with this last-mentioned group of cases it is well to remark that high sphygmometer readings, and even very high readings, by no means necessarily involve retinal haemorrhage; indeed, in the majority of cases of this order the fundus oculi is normal, or at least free from haemorrhage;

and this is true even when so disastrous an event as cerebral apoplexy is the commanding fact in the clinical picture. Thus it seems safe to say that retinal haemorrhages are not produced by the "bursting" of healthy blood vessels—or for the matter of that of degenerated blood vessels—under the stress of increased intravascular pressure, even allowing—that is a disputed point—that a high reading obtained from the brachial artery means increased blood pressure within the retinal vessels. Retinal haemorrhages, as I have already argued, mean, indeed, rupture of vessels, but the essential cause of this rupture is, not increased intravascular stress, but degenerative changes in the vessel walls. Support for these propositions is found in yet another group of cases—namely, those in which there is manifest arterial degeneration without high sphygmometer readings; for in such cases retinal haemorrhages may be present—may be present, that is, when, at least so far as the sphygmometer can be trusted, the blood pressure is not abnormal. In other words, high sphygmometer readings—or, if you will, high blood pressures—do not *per se* involve vascular rupture, while vascular disease may lead to rupture even when the alleged measurement of blood pressure is well within normal limits. It is change in the value of the vessel wall that is the necessary preliminary to rupture and haemorrhage, and this change either may or may not be associated with a high sphygmometer record.

Perhaps here I may be allowed a word in appreciation of the work done by ophthalmic surgeons on the pathology and clinical evidences of degenerative disease of the blood vessels. It has helped towards the solution of not a few obscure problems.

Of retinal haemorrhages in glycosuria there is little to be said. They are found, as everyone agrees, not in the typical diabetes of the earlier years of life but in middle-aged or elderly persons with slight or moderate symptoms and non-extreme degrees of glycosuria. They may or may not be accompanied by retinitis. When, as sometimes happens, the glycosuria disappears for a more or less prolonged interval the ophthalmoscopic picture may be a very puzzling one for an observer ignorant of the patient's earlier history. Upon prognosis in glycosuria retinal haemorrhage as such exerts little or no influence; the haemorrhage is quite consistent with active life continued during many years.

In the course of our discussion we are likely to hear of the differences—the diagnostic differences, as some affirm—between retinal haemorrhages in glycosuria and retinal haemorrhages in albuminuria. I admit the interest of the comparison as a subject for ophthalmoscopic contemplation and as an opportunity for the cultivation of the sporting spirit in the art of diagnostic adventure. But the observation has little clinical value. At best the conclusion suggested is a probability, and a probability which no one will say is invariably justified by the event. Moreover, the procedure is an offence against a sound and sensible rule—important in medicine as in law—that second-rate evidence ought not to be accepted when first-rate evidence is available.

I have now briefly to refer to certain clinical situations in which the discovery of retinal haemorrhages has a high practical value, inasmuch as such a discovery may save the practitioner from error and may indicate the route along which an accurate diagnostic conclusion is to be sought. Experiences of retinal haemorrhages as the first clinical event in renal disease, in glycosuria, and in arterial degeneration, are obvious illustrations of this proposition. A few others may be mentioned.

First, a doubtful or ambiguous appearance at the optic disc will be confidently interpreted if the suggestion of a possible commencing optic neuritis is accompanied by one or more small linear haemorrhages. They may require close observation, but once detected there can be no question of their significance.

Next I mention septicaemia and septic endocarditis. Quite a number of such cases are clinically, and for a more or less prolonged period, cases of unexplained temperature—fever without physical signs; and such diagnoses as tubercle, enteric fever, paratyphoid, and *Bacillus coli* infection of the urinary passages come up for discussion; or, again, with the fever are joint pains, and acute rheumatism offers itself as an appropriate label; and once more, in a patient known to be the subject of a chronic valvular lesion occurs an outbreak of febrile temperatures, and a recrudescence of active rheumatism is a tempting explanation. Yet in every one of these instances an ophthalmoscopic examination may promptly and confidently alter both the diagnostic position and the prognostic

outlook, for retinal haemorrhages are not to be expected either in tuberculosis, or in enteric fever, or in paratyphoid, or in *B. coli* infections, while in septicaemia and in septic endocarditis they are quite frequent events.

Another disease in which the presence of retinal haemorrhages affords much help is pernicious anaemia. Here again, though rarely I admit, the problem presents itself in the form of unexplained pyrexia, and the anaemia and weakness are readily regarded as consequences of the high temperature, and for this an explanation is sought in the directions already indicated; the detection of retinal haemorrhages in such circumstances is of great practical value. On the other hand, a more or less severe and prolonged anaemia may be attributed to chlorosis, to dyspepsia, to malnutrition, or to some other more or less indefinite influence. The recognition of retinal haemorrhages here ought to sound a note of alarm, for such haemorrhages are frequent in pernicious anaemia, whereas in chlorosis and in secondary anaemias they are either absent or, if occasionally present, are slight in degree, few in number, and confined to extreme and advanced cases. Even a wider generalization may be ventured—namely, that retinal haemorrhages in the absence of renal disease, of glycosuria, and of arterial degeneration mean either sepsis or one of the so-called blood diseases, and particularly pernicious anaemia and leukaemia. Rarely they are due to malaria, and very rarely to syphilis; personally, I have not seen them in haemophilia or in Hodgkin's disease, and only once in purpura haemorrhagica. Retinal embolism and thrombosis of the retinal vessels perhaps ought to be mentioned, but in each of these there are in the history and in the ophthalmoscopic examination characteristic facts which reduce the attendant haemorrhages to a purely subordinate position. In addition, the generalization just proposed is qualified by the "unexplained" haemorrhages to which reference has already been made, and by the necessary remark that propositions which pretend to absolute values in the field of clinical medicine, however serviceable for general guidance, are apt now and again to suffer in their dignity by the arrival of events and combinations which contradict them.

In a congress of ophthalmic surgeons it would be gratuitous to insist on the importance of examining each fundus oculi, and yet it may be well to remark that even in so generalized a disease as pernicious anaemia retinal haemorrhages may have, at any rate for a time, a purely unilateral distribution.

CONCLUSIONS.

Finally, I submit the following practical conclusions—namely:

1. That retinal haemorrhages may exist without recognized prejudice to vision, and ophthalmoscopic examination is therefore a necessary part of every clinical examination.
2. That such haemorrhages may be the first objective signs of serious disease, and a discovery of them, therefore, demands a complete examination of the patient.
3. That the recognition of retinal haemorrhage is often of high value in directing the observer to a correct interpretation of the clinical facts, while the prognostic significance of the observation *per se* is indeterminate.
4. That retinal haemorrhages do occasionally exist as isolated clinical facts, and when so existing are comparable to haemorrhages in other parts of the body (haematemesis, haematuria, haemoptysis, etc.), for which no ready explanation is at hand.

A METHOD OF REDUCING CONGENITAL DISLOCATION OF THE HIP.

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REDUCTION of a congenital dislocation, except in very young children, is not always the simple matter described in the textbooks. When the ordinary methods of pulling, lifting, and levering are employed the operator works under mechanical difficulties, and, unless well equipped physically, is likely to fail. So much energy is expended in pulling that proper control over the movements of the head of the femur is lost, and if the help of an assistant be utilized there is often want of complete co-ordination between the manipulators. In any case much energy is wasted. The method here described appears to me to avoid misdirected effort and